ISSN 1990-7478, Biochemistry (Moscow), Supplement Series A: Membrane and Cell Biology, 2017, Vol. 11, No. 4, pp. 253–260. © Pleiades Publishing, Ltd., 2017.
Original Russian Text © S.N. Grishin, A.I. Gabdrakhmanov, A.E. Khairullin, A.U. Ziganshin, 2017, published in Biologicheskie Membrany, 2017, Vol. 34, No. 4, pp. 251–260.

REVIEWS

The Influence of Glucocorticoids and Catecholamines on the Neuromuscular Transmission

S. N. Grishin[a], A. I. Gabdrakhmanov[b], A. E. Khairullin[b, d], and A. U. Ziganshin[a, c, *]

[a]*Kazan State Medical University, Department of Medical and Biological Physics with Computer Science and Medical Equipment, Kazan, 420012 Russia*

[b]*Kazan Federal (Volga Region) University, Kazan, Institute of Fundamental Medicine and Biology, Kazan, 420008 Russia*

[c]*Kazan State Medical University, Department of Pharmacology of Pharmaceutical Faculty, Kazan, 420012 Russia*

[d]*Kazan State Medical University, Department of Biochemistry, Kazan, 420012 Russia*

**e-mail: auziganshin@gmail.com*

Received October 1, 2016; in final form, January 7, 2017

Abstract—The review surveys the impact of "stress hormones"–glucocorticoids and catecholamines–on the functioning of the neuromuscular synapse. The review brings together the data on the influence of the main agents of stress – cortisol and norepinephrine–on the intensity and timing of the acetylcholine release, as well as signaling effect of its co-mediator ATP.

Keywords: stress hormones, neuromuscular synapse, catecholamines, glucocorticoids, muscle contraction, cortisol, noradrenaline, synaptic modulators

DOI: 10.1134/S1990747817040043

INTRODUCTION

The state of stress appears in an organism exposed to supernormal, right up to extreme and damaging, stimuli [1–3]. Under the influence of extreme factors, nonspecific stress reactions of an organism are aimed primarily at mobilization of energy for adaptation processes [2]. Glucocorticosteroids and catecholamines released into blood play the key role in these nonspecific reactions. By activating catabolic processes, these agents cause hyperglycemia and hyperlipidemia—the initial reactions of substrate energy supply, which are also needed to enhance the efficiency of the locomotor system, the basic mission of which under stress is avoidance of or active resistance to supernormal stimuli [3].

Alternatively, now we have acquired more insight into existence of a complex pool of targets of the key signaling molecules. For example, it has been revealed that the agents with action previously considered to be strictly central exert influence on neuromuscular junctions [4–6].

The study of the effects of stress hormones on the function of neuromuscular junction was started in the middle of the past century; however, the numerous published results are often contradictory [7–14]. In the most recent reviews [15], the very possibility of the effect of catecholamines on neuromuscular transmission is doubted, i.e., inter alia, their previously revealed involvement in the "Orbeli-Ginetsinski phenomenon" is denied [16].

This review surveys the data on the effects of catecholamines and glucocorticoids on neuromuscular transmission.

GLUCOCORTICOIDS

Glucocorticoids are steroid hormones produced by the adrenal cortex [17]; the major and most active of them in humans is cortisol. The blood level of glucocorticoids dramatically increases in the case of stress, shock states, injuries, and blood loss. This reaction is considered to be the key mechanism of adaptation of an organism to acute stress [17, 18].

Glucocorticoids influence the functional state of neuromuscular junction by acting on the protein-synthesizing apparatus and changing the DNA-dependent synthesis of RNA [19, 20].

Previously it was believed that steroid hormone receptors were present only in the cell nucleus and that the hormone–receptor complexes were transcription factors [21]. Then it became clear that glucocorticoid receptors are also present in the cell membrane [21–23].

It was shown that glucocorticoids exert some immediate effects not depending on the gene transcription regulation. The binding of glucocorticoids with the glucocorticoid receptor simulates phosphati-